ANÆSTHESIA FOR MITRAL VALVOTOMY*

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The object of this paper is to discuss the use of nitrous oxide supplemented with pethidine in the operation of mitral valvotomy for the relief of mitral stenosis.

Direct surgery of the stenosed mitral valve is no new conception. Indeed, as far back as the year 1902, Brunton put forward the plea for surgical disruption of the stenosed valve, but he met with such determined opposition that his idea was allowed to lapse into obscurity. It was H. S. Souttar of the London Hospital who, in 1925, performed the first mitral valvotomy on a girl of 15 years of age. Anaesthesia in this case was punctuated by a series of crises, the pulse rate rising at times to 150 per minute and the heart being grossly irregular on handling. Despite an immediate post-operative systolic blood pressure of 60 mm. Hg, the patient made an uneventful recovery. Pribram, in Germany, reported one successful case in 1928.

It is to Bailey of Philadelphia that we owe the modern operation of mitral commissurotomy, that is the splitting of the stenosed valve at either commissure. Following the publication of his work in 1949 many surgeons have attempted the operation. Baker, Brock and Campbell (1950), Brock (1950), in London; Murray (1950), in Montreal and Smithy, Boone and Stallworth (1950) in Baltimore, have published several papers on this operation.

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At this junction I should like to consider for a few moments the patient with mitral stenosis. Mitral stenosis follows, often after a long period, rheumatic infection of the heart. Although this may have occurred in youth or early adult life, the rheumatic infection may have passed unnoticed. In fact many patients with severe stenosis have no previous history of rheumatic disease.

Dyspnoea is one of the earliest symptoms of mitral stenosis and this is noticed at first only after fairly severe exercise. However, the onset of dyspnoea occurs earlier as the stenosis becomes more severe.

In figure 1 I have plotted, diagrammatically, exercise tolerance against time in years, the years across which the disease pursues its relentless path. Exercise tolerance is adjudged as the point at which dyspnoea results in the performance of standard graded exercises, such as walking on the level, or up a series of steps. It is surprising how little a patient with severe stenosis will be able to perform. It will be seen from the figure that as the disease progresses so the exercise tolerance diminishes.

The left auricle dilates and hypertrophies and, due to damming back of blood in the pulmonary system, the right ventricle in turn becomes dilated and hypertrophied with a consequent rise in the pulmonary artery pressures. This in turn results in chronic cough with a marked tendency to attacks of pulmonary oedema. Indeed, in the later stages of the disease, attacks of pulmonary oedema may be frequent and may result in right-sided cardiac failure and death.

Chronic venous congestion also becomes apparent, the systemic veins become dilated, the liver enlarged and peripheral oedema, especially of the ankles and sacral region may be seen. Cyanosis is often severe and the typical
mitral facies is frequently seen. Clubbing of the fingers is also a fairly common finding. Auscultation of the chest nearly always reveals moist sounds at both bases and the patient may have in addition a chronic bronchitis. Nocturnal dyspnoea is a common finding and the patient may frequently sleep in the sitting position.
In the later stages of the disease frequent small hae-moptyses occur and are a further sign of a rising pulmonary artery pressure.

Sooner or later, evidence of myocardial damage will be seen electrocardiographically and auricular fibrillation and flutter are common. It is possible to perform the operation at all stages of disease, even in the case with frank auricular fibrillation. Several of the cases in this series were in fact fibrillating before, during and after valvotomy.

THE OPERATION

Briefly the operation consists of a left thoracotomy with, usually, resection of the 4th rib. The left lung is collapsed and the pericardium incised widely, anterior to the phrenic nerve.

The left auricle is entered from the left auricular appendage and the surgeon's index finger is inserted through the appendage and into the auricle, the mitral valve being split at either commissure. In figure 2 the finger is seen passing through the left auricle and the mitral valve. The finger is then withdrawn, a special clamp is applied to the base of the appendage, and the latter is amputated after suture of its base. The pericardium and thorax are then closed in layers, the left lung being fully re-expanded before closure of the chest and a water-sealed drain left in place.

ANAESTHESIA

I regard the following as the basic essentials in any anaesthetic technique for the operation of mitral valvotomy.

(1) Adequate oxygenation of the patient at all times.
(2) Complete control of respiration.
(3) Reduction of cardiac irritability.
Fig. 2
Diagrammatic representation of the surgeon's finger in the mitral valve. The wall of the left ventricle has been resected.
(4) The Anaesthetic Drugs of choice should be non-irritable to the myocardium, non-toxic to other tissues, preferably be non-inflammable, and to allow of a quick recovery rate with minimal post-operative sequelae.

(1) Adequate oxygenation. Adequate oxygenation, essential at all times during anaesthesia, is vitally so during intra-cardiac surgery. The heart in mitral stenosis is already working under a severe handicap and, if the oxygen content of the blood is allowed to fall, serious results will ensue, both as regards the myocardium and the cerebral tissues.

(2) Complete control of respiration is obtained by the use of d-tubocurarine chloride and voluntary respiration is further prohibited by the use of a Waters' to-and-fro absorber connected to a cuffed endotracheal tube. In the more recent cases use has been made of the Blease Pulmoflator, with great success. This apparatus administers any previously determined tidal volume, the intra-pulmonary pressure can be set and the rate and length of inspiration and expiration adjusted at will. The Pulmoflator is undoubtedly a great asset to the single-handed anaesthetist in intra-cardiac or thoracic surgery.

(3) Irritability of the myocardium. The myocardium resents handling and reacts to it with many varied arrhythmias. These may vary from the simple auricular extrasystole to frank auricular fibrillation. Partial or complete heart block may be seen, or varied ventricular extrasystoles heralding the onset of ventricular fibrillation, and sudden death may occur.

It has been known since 1942, when Lundy discussed the use of intravenous procaine, that this drug possessed a quinidine-like action on the myocardium, in that it pro-
longed the refractory period and at the same time reduced the conductivity of the conducting bundles. Burstein, in 1946, described its use in controlling cardiac irregularities during anaesthesia.

In the earlier cases of the series a 0.1 per cent solution of procaine hydrochloride in saline was used, but, as it was felt that the use of such quantities of intravenous saline might induce the onset of pulmonary oedema, the use of procaine in this form has been discontinued. Moreover, it is known that procaine hydrochloride is rapidly and completely destroyed in the blood plasma by the enzyme procaine-esterase.

Procaine amide, like procaine hydrochloride, depresses the irritability of the ventricular muscle. Unlike the latter, however, it is only partially hydrolysed by procaine-esterase so that its effect is much more prolonged. Its action occurs immediately after injection intravenously and the plasma level declines by about 10 to 15 per cent per hour.

Clinically the amide will abolish ventricular ectopic beats, paroxysmal ventricular tachycardia and ventricular extrasystoles, but has no effect on persistent auricular fibrillation or flutter. To correct ventricular arrhythmias during anaesthesia, procaine amide is given intravenously in a dosage of 100 mg. to 500 mg. (1 to 5 ml. of the 10 per cent solution).

The commonest side effect is hypotension and for this reason the drug should be given in divided doses. The usual practice is to give a test dose of 100 mg. and continue carefully with repeated increments of 100 mg., keeping a careful check on the blood pressure.

(4) Anaesthetic drugs of choice. Cyclopropane is relatively non-toxic to the tissues and allows an adequate blood oxygenation. The recovery period though rapid is not so rapid as with nitrous oxide, and I believe that there is an
increased incidence of post-operative vomiting and nausea with the former. At the same time cyclopropane can produce many undesirable arrhythmias and this is to be avoided at all costs in these cases. The explosive qualities of cyclopropane, moreover, forbids the use of diathermy inside the chest, and there seems to be little doubt that capillary oozing is increased with this agent.

Ether, similarly, is an explosive drug, and although relatively non-toxic to the myocardium and other tissues, the recovery period is slow and often accompanied by nausea, vomiting and possibly post-operative chest complications. Capillary oozing is similarly increased.

I consider that nitrous oxide in a 50 per cent mixture with oxygen, supplemented with intravenous pethidine hydrochloride is the anesthetic drug of choice. Nitrous oxide is non-toxic to the myocardium in this percentage, is non-inflammable, allowing the use of diathermy inside the chest, recovery is rapid and post-operative sequelae are minimal. The absence of post-operative vomiting in this series has been most striking. Pethidine hydrochloride gives adequate analgesic supplementation allowing a 50 per cent N₂O:O₂ mixture to be used and has a prolonged analgesic effect post-operatively. At the same time pethidine has, according to Burn (1950), a quinidine-like action on the heart in that it prolongs the refractory period. The drug, therefore, has a useful place in the realms of intracardiac surgery.

**Technique employed**

The premedication employed is papaveretum and scopolamine, the maximum dose being papaveretum 1/3 grain (20 mg.) and scopolamine 1/150 grain (0.45 mg.) given some 1½ to 1½ hours pre-operatively by subcutaneous injection.
When the patient is brought to the anaesthetic room, a cannula is inserted, under local anaesthesia, into the right internal saphenous vein at the ankle. An immediate slow blood drip is set up.

Anaesthesia is induced by the injection of up to 250 mg. thiopentone into the drip, followed by 15–20 mg. d-tubocurarine chloride which is then followed by another 100–250 mg. thiopentone. These doses, however, are adjusted according to the age and condition of the patient.

The lungs are then rhythmically inflated with oxygen for a period of two minutes in order to allow maximum jaw relaxation. The patient is intubated with the largestuffed endotracheal tube that can be passed without trauma to the cords. The cords are not sprayed routinely with analgesic solutions.

It is important to check the position of the tip of the endotracheal tube, and inspection and auscultation of the chest must be routine in order to avoid the possibility of the tube having entered a main-stem bronchus. Controlled respiration with a 50 per cent N₂O:O₂ mixture is now commenced, and the patient is taken into the theatre.

The patient is now set up on the table in the lateral position, left side uppermost (fig. 3). The left arm is suspended from an arm rest, the right arm being placed beneath the patient's head with the arm fully flexed at the elbow.

A test dose of 25–50 mg. of pethidine is given shortly after induction and supplementary doses of 25 mg. pethidine and/or 5 mg. d-tubocurarine are injected into the drip as required. Slight voluntary movements, such as twitching of the fingers or eyebrows, indicate the need for a further increment of pethidine. A rising pulse rate similarly indicates the need for further pethidine, but in this operation this is not so reliable a sign, as the pulse may swing
Patient is shown in position on the operating table. The leads for the electrocardiograph can be clearly seen.
Fig. 4
View of head end of table to show Blease PulmoIiator and electrocardiograph in use during operation.
from time to time for other reasons. The Blease Pulmoflator is now connected up and the patient is automatically inflated 18–20 times per minute at a tidal volume previously determined (fig. 4). Usually this is about 500 ml.

Up to the exposure of the left auricle 100–300 mg. procaine amide is administered intravenously, depending on the state of irritability of the heart on direct handling. This is determined most easily by direct vision, but continuous electrocardiographic tracings, from a portable electrocardiograph are taken routinely. Lead II tracings are the most informative. An extra damping effect can be obtained at any stage by increasing the dosage of procaine amide and asking the surgeon to desist for a few minutes to allow the heart to settle. Handling of the left auricle should, if sufficient amide has been given, present little in the way of irregularities in rhythm or rate.

The blood pressure falls slightly following disruption of the valve (fig. 5) but should return to normal levels fairly quickly. The pulse rate shows no significant changes as a rule. Multiple ventricular extrasystoles are seen during the actual disruption of the valve, but there is usually an immediate return to normal sinus rhythm.

Closure of the pericardium may in some cases produce a marked bradycardia which is suggestive of vagal inhibition, but this can be quickly corrected by the administration of atropine intravenously (fig. 6).

As soon as the base of the appendage has been sutured a definite attempt should be made to inflate the lung and the surgeon must be requested to desist from the operation at this stage. It is important that all lung lobules are fully inflated, as it may be found impossible to do this at the end of the operation. In fact it is of great advantage to the patient to inflate the lung several times during the course
Fig. 5
Figure to show slight fall in blood pressure following disruption of the mitral valve

Fig. 6
Showing the effect of intravenous atropine in a case of bradycardia following closure of the pericardium
of the operation. On closure of the chest the lung must be fully inflated. An intercostal drain is led under a water-seal, any air which is left in the pleural cavity being displaced by inflation of the lung.

On removal of the absorber spontaneous respiration returns, and the operation is completed by a thorough toilet of trachea and bronchi. Much importance is attached to this procedure as the patient must not be returned to the ward with his trachea and bronchi full of secretions. Bronchoscopy should be performed if there is any doubt regarding fluid in the bronchial tree.

In the ward the patient is placed in an oxygen tent through which a flow of 6–8 litres of oxygen per minute is run. A careful watch is kept on pulse rate, colour, blood pressure and respiration. Little sedation is required at first, but pethidine usually gives adequate analgesia.

In the most recent cases, in an attempt to cut down post-operative chest pain, patients have been given an intercostal block, above and below the wound to the extent of two spaces with 1 to 2 ml. of Efocaine in each intercostal space. This drug will, in the average case, produce analgesia lasting from 8 to 14 days. So far in the limited number of cases so treated, the results have been most gratifying, as the patient is able to take deep breaths without chest pain right from the first post-operative day.

It is surprising how well these cases look post-operatively, and they can be seen within a few hours sitting propped up in bed inside their oxygen tents. The oxygen tent is usually dispensed with by the second day, unless there is some residual cyanosis.

RESULTS

To date I have administered anaesthesia to some 33 of these cases. On the debit side two cases unfortunately died in the post-operative period.
Complications during operation

In the first case, a male aged 37 years, a large number of organized thrombi were discovered on opening the auricular appendage. In order to prevent their reaching the cerebral circulation the surgeon asked me to compress both common carotids. This was done with the result that the heart ceased to contract for 12 seconds (fig. 7) and on

![Graph showing blood pressure and pulse rate over time.](image)

**Fig. 7**  
Case in which the heart stopped for twelve seconds following compression of both carotid arteries.

resumption of contraction numerous bursts of ventricular extrasystoles were seen. Two ml. of 2 per cent procaine and 1/1000 adrenaline were injected into the left ventricle with a gradual return to normal rhythm. This was undoubtedly a case of vagal inhibition following stimula-
tion of the carotid body reflex. This could have been prevented by the administration of atropine before compression, but in the emergency no time was allowed for this.

The patient recovered subsequently, but commenced to fibrillate on the third post-operative day. Cyanosis and dyspnoea were present and both increased. Large pleural effusions formed, especially on the left side, one litre of fluid being aspirated. Death occurred on the tenth post-operative day due to progressive cardiac failure. Autopsy revealed a mitral valve which had only been split very slightly at one commissure.

During the second case, a female of 38 years, the surgeon unfortunately lost control of the auricular opening during the actual splitting of the valve. There was a sudden loss of some 1½ pints of blood. The blood pressure fell to unrecordable depths, the heart being grossly irregular with multifocal ventricular extrasystoles. The patient, however, improved, but unfortunately died on the second post-operative day due to cardiac failure. There was massive bilateral pleural effusions and examination of the valve revealed a tiny split at one commissure only.

Post-operative complications

As regards post-operative complications, table I shows the main complications in the 32 cases.

One of the commonest has been that of auricular fibrillation and this has occurred most frequently from the third to the fifth day. Ten cases showed fibrillation in this period, but of these, five were fibrillating before, during and after operation. Routine quinidine administration from the first post-operative day seems to a large extent to control most of these potential fibrillators, but again some respond better to digitalis and digoxin.

The next commonest complication has been that of
TABLE I
Post-operative Complications in 33 Cases

1. **Cardiac Irregularities**:
   (A) Auricular fibrillation ... ... 10 (5)
   (B) Partial heart block ... ... 1

2. **Chest Complications**:
   (A) Atelectasis:
     - left lower lobe ... 2
     - other lobes ... 2
   (B) Pleural effusions:
     - large bilateral ... 2
     - small left basal ... 6
   (C) Pericarditis with effusion ... ... 1
   (D) Dyspnoea (undiagnosed) ... ... 1

3. **Other complications**:
   (A) Vomiting ... ... ... ... 3
   (B) Thrombosis (calf veins) ... ... 1
   (C) Nerve palsy ... ... ... ... 1
   (D) Mental changes ... ... ... ... 1
   (E) Cyanosis ... ... ... ... 2

collapse of the left lower lobe with or without small left basal effusions. Adequate re-expansion of the lung at frequent intervals during the operation seems to be the answer as regards the lower lobe collapses, and maintenance of a patent drainage tube should prevent the collection of basal effusions.

One case of pericarditis with effusion occurred, but this was undoubtedly due to the use of 4 per cent procaine inside the pericardial sac, a procedure now abandoned.

Vomiting in this series has been seen only on rare occasions. In three cases vomiting was severe, and was undoubtedly due to overdosage with digitalis, as on the withdrawal of this drug vomiting ceased.

A temporary paralysis of C5 and C6 roots on the left side occurred in one patient due to faulty placing of the left arm, but happily complete recovery took place after three months.
In another case severe mental changes of an emotional character took place after the operation. As there was no evidence of motor loss, the presence of a cerebral embolus can only be conjectured, but the surgeon reported the presence of several emboli in the left auricular appendage at operation, and it is possible that one may have entered the cerebral circulation. Further, compression of both carotid arteries in this case may have caused some permanent cerebral damage from anoxia of brain cells.

As regards post-operative sedation, I was surprised to find that the average amount of pethidine used post-operatively over the first ten days was in the region of 1,800 mg. Since performing intercostal blockade of the wound with Efocaine this figure has been reduced to 900 mg. This is undoubtedly a significant drop. Pethidine is not administered routinely but only when the patient really requires relief of pain. There is no doubt that a properly performed intercostal block with Efocaine will cut down post-operative chest pain, and at the same time reduce the amount of sedative required in this period.

SUMMARY OF ANAESTHETIC TECHNIQUE

In conclusion I should like to summarize the anaesthetic technique I have employed in the operation of mitral valvotomy.

1. Induction. Thiopentone.

   Maintenance. 50 per cent N₂O: O₂,+pethidine.

2. Adequate oxygenation throughout operation.

3. The use of curare and controlled respiration.

4. Reduction of myocardial irritability with procaine amide.

5. Average total dosage of anaesthetic drugs used in 32 cases:
Finally, I should like to thank Mr. W. Arthur Mackay and Mr. Kenneth Fraser for permission to publish their cases, also I am indebted to Dr. A. G. Miller and Dr. H. H. Pinkerton for much help and useful criticism in the preparation of this paper.

REFERENCES